AMENDMENT

Kindly amend the specification, without prejudice, without admission, without surrender of subject matter, and without any intention of creating any estoppel as to equivalents, as follows.

IN THE CLAIMS:

Kindly amend the claims, without prejudice, without admission, without surrender of subject matter, and without any intention of creating any estoppel as to equivalents, to read as follows:

- 1. (Previously presented) A transgenic mouse model showing hypomyelinosis of the thalamus wherein the transgenic mouse comprises a homozygous disruption in chromosomal DAP12 (DNAX Activation Protein 12) gene function, and wherein the homozygous disruption includes the promoter region and exons 1, 2, and 3.
 - 2. (Canceled)

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- 3. (Currently amended) The transgenic mouse model of claim 1, wherein the homozygous disruption in DAP12 can be pheonotypically exhibited as a myelinogenesis developmental disorder or a neuropsychiatric disorder associated with disruption in DAP12 gene function.
- 4.(Currently amended) The transgenic mouse model of claim 3, wherein the neuropsychiatric disorder is selected from the group consisting of Nasu-Hakola disease, dementia associated with disruption in DAP12 gene function, schizophrenia associated with disruption in DAP12 gene function, schizotypal personality disorders associated with disruption in DAP12 gene function, obsessive-compulsive disorders associated with disruption in DAP12 gene function, or Tourette's syndrome associated with disruption in DAP12 gene function.
- 5. (Currently amended) The transgenic mouse model of claim 3, wherein the neuropsychiatric disorder is Nasu-Hakola disease or dementia <u>associated with disruption in DAP12</u> gene function.
 - 6-18. (Canceled)

Page 2 00421539

- 19. (Previously presented) The transgenic mouse model of claim 1, wherein the expression of myelin basic protein in the brain is weak in regions where DAP12 is strongly expressed in wild-type mice.
- 20. (Previously presented) The transgenic mouse model of claim 1, wherein the transgenic mouse exhibits an impairment in sensorimotor gating as compared to wild-type mice.

Page 3 00421539